Tobacco Smoke Constituents

Tobacco smoke is a highly complex mixture of thousands of ingredients, whose effects, if any, on human health have not been scientifically established. Despite this, claims are frequently made that tobacco smoke or one of its constituents (usually condensate, nicotine or carbon monoxide) causes human disease. Such claims are largely based on two types of studies -- epidemiological and laboratory. The former are population studies in which smoking has been reported to be statistically associated with certain diseases. However, scientists generally agree that statistical associations cannot establish causal relationships. As a distinguished medical statistician noted, "cancer is a biologic, not a statistical, problem."[1]

The latter group of studies report findings from laboratory experiments using extremely large amounts of tobacco smoke or one of its constituents under unrealistic test conditions. As a result of these conditions, caution must be used in applying the conclusions of such studies to humans. In addition, the results of such studies have not been consistent; consequently, serious questions about their meaning can be raised.

Therefore, in the words of a scientist, "after years and years of intensive research, no compound, as found in cigarette smoke, has been established as a health hazard."[2]

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Condensate ("Tar")

There is no such constituent as condensate (or "tar" as it is commonly called) in cigarette smoke. That is why the term "tar" is frequently placed in quotation marks. The substance called "tar" is actually produced in the laboratory by collecting the particulate matter in tobacco smoke. The collection method most often used involves passing the smoke through a cold trap at extremely low temperatures. As a United States government report on smoking and health acknowledges, such mechanical processes hardly duplicate the way humans smoke:

> There is, at present, no available instrumentation permitting the separation and individual collection of the particulate and gas phases which duplicates the precise physiochemical conditions prevailing in cigarette smoke as it is inhaled.[3]

Nor is it clear that the "tar" collected in this way is necessarily the same as the substance which is eventually used in animal experiments. That is because "tar" continues to undergo changes while it is being stored for study.[4] Even the 1979 U. S. Surgeon General's Report noted that "the smoke aerosol [from which the "tar" is collected] is a continuously changing entity. Aging of the aerosol results in changes in its physical and chemical properties."[5]

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Despite the fact that "tar" is not found in tobacco smoke, this substance has been used in animal experiments which purportedly examine the relationship between smoking and cancer. In many of these experiments, "tar" was repeatedly painted on the skin of test animals over long periods of time. Tumors which may have developed were then said to be caused by the "tar".

However, the relevance of these experiments to the human experience has been questioned. For example, there are "many important differences" between animal skin and human lung tissues.[6] Moreover, the amounts of "tar" used in these experiments have often been incredibly high. In one situation, it was estimated to be the equivalent of smoking 100,000 cigarettes per day.[7] A researcher commenting on these experiments contended they involve the application of "the wrong material, in the wrong form, in the wrong concentration, to the wrong tissue of the wrong animal."[8]

For such reasons, a scientist has concluded that "human beings do not smoke 'tar' and laboratory reports on 'tar' yields have not been established as significant to human health."[9]

Nicotine

One of the most comprehensive reviews of tobacco smoke components was conducted during the preparation of the

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1964 U. S. Surgeon General's (Terry) Report. In discussing nicotine, the report noted that the amount absorbed from tobacco smoke "is very low and probably does not represent a significant health problem."[10] The most recent review of this topic by the U. S. Surgeon General in the 1979 report indicates that no data have been collected since that time which would require a change in this statement.[11]

Regardless of these observations, nicotine has been blamed for playing a role in the causation of heart disease by clogging the arteries (arteriosclerosis). A German researcher concedes this is "very unlikely," however, based on his analysis of animal studies designed to study the possible effects of nicotine administration.[12] Despite the "very large doses of nicotine" used in these experiments, which he calculated to be the equivalent of smoking up to 1,200 cigarettes per day, the researcher found that nicotine, "when administered alone, has no injuring effect" on the interior walls of the blood vessels.[13] The excessively high amounts of nicotine used in such animal experiments have been criticized by other researchers:

> There have been some studies which have exhibited minor or questionable changes with the use of an equivalent dose of 600 or more cigarettes a day in man. This is such a large number that I think man would find it difficult to find the time to smoke them.[14]

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In view of the observations by the German researcher, it should not be surprising that animal experiments using more realistic doses of nicotine have also failed to show any influence on the arteriosclerotic process:

> In these experiments it was clearly demonstrated in the rabbit that realistic doses of either nicotine or cigarette smoke failed to initiate, exacerbate, or otherwise influence the atherogenic process in that species.[15]

In its evaluation of such experiments, the 1979 U. S. Surgeon General's Report observed that "the overall impression from available data is that nicotine does not affect atherogenesis in animals."[16]

Accordingly, a scientist who has reviewed research in this area concluded that "it has never been scientifically established that nicotine causes or contributes to atherosclerosis or -- for that matter -- any other cardiovascular disease."[17]

Carbon Monoxide (CO)

Despite the limited number of studies in humans examining the possible relationship between CO and the development of cardiovascular conditions, it has been claimed that CO in cigarette smoke causes heart disease. However,

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these claims are not supported by studies of tunnel workers and motor vehicle examiners exposed for many years to unusually large amounts of CO. In these studies, workers were not found to have higher rates of cardiovascular disease than the general public.[18]

Perhaps because these studies have failed to prove their point, anti-smoking advocates have emphasized the results of certain animal studies, particularly those conducted by a team of Danish scientists. In their studies, the Danes reportedly had induced vascular changes in rabbits similar to early atherosclerosis in man by exposing the animals to large quantities of CO and a high cholesterol diet.[19] Recently, however, these same researchers announced that they have been unable to duplicate their earlier results.[20] According to one of the researchers:

> An atherogenic or histotoxic effect of carbon monoxide (CO) could not be demonstrated in coronary arteries, aorta, lung tissue, or pulmonary vascular system, following exposure to low and high level CO in air for various periods of time.[21]

In its discussion of these and other animal studies, the 1979 U. S. Surgeon General's Report stated that they "have produced conflicting results and are inconclusive."[22]

A German scientist who is known for his anti-smoking views also recently noted that "evidence linking CO as the causative agent [to CVD] has not been established."[23]

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Still other claims have been made that CO from cigarette smoke adversely affects the smoker's ability to perform tasks, such as judging time intervals and making decisions associated with driving an automobile. However, respected researchers who have conducted experiments in which individuals were exposed to CO have found that the smokers' performance ability is not affected by long-term exposure to CO.[24]

In a recent study on performance, in fact, a group of researchers was unable to reproduce the findings of previous studies which have been cited as showing performance decrements in subjects exposed to CO.[25] In their discussion of such studies, the researchers concluded that the data concerning such effects are "inconclusive."[26] Similarly, a review of studies examining the alleged effects of CO on driving performance expressed concern about "the lack of clear-cut evidence" in this area.[27]

A U. S. federal agency also addressed this issue when it denied the petition of several anti-smoking groups who had sought to prohibit smoking by airline pilots in the cockpits of commercial aircraft.[28] Government scientists found no impairment in performance either at the low levels of exposure that occur when tobacco is smoked or at much

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higher exposures. In its decision, the agency stated that the existing data are "too inconclusive to warrant the issuance of the requested rule at this time."[29]

Obviously, many questions remain unanswered about what role, if any, tobacco smoke and its components play in disease causation. A researcher who commented on such questions asserted that "neither these epidemiological studies, nor animal and clinical studies, have identified any ingredient or group of ingredients as found in smoke as disease producing in humans."[30] These words add support to the conclusion that claims about the harmful effects of tobacco smoke on health have not been scientifically established.

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